

# WEST Search History

DATE: Thursday, February 05, 2004

<b>Hide?</b>	<b><u>Set Name</u></b>	<b><u>Query</u></b>	<b><u>Hit Count</u></b>
		<i>DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L2	(L1 or pacap) and @pd > 20040113	15
<input type="checkbox"/>	L1	(pituitary adenylate cyclase activating polypeptide) and @pd > 20040113	6

END OF SEARCH HISTORY

\$%^STN;HighlightOn= \*\*\*;HighlightOff=\*\*\* ;

)

=> d bib abs

Welcome to STN International! Enter xx

LOGINID:ssspta1633coq

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR 7):2

\*\*\*\*\* Welcome to STN International \*\*\*\*\*

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 "Ask CAS" for self-help around the clock  
NEWS 3 SEP 09 CACApplus records now contain indexing from 1907 to the present  
NEWS 4 DEC 08 INPADOC: Legal Status data reloaded  
NEWS 5 SEP 29 DISSABS now available on STN  
NEWS 6 OCT 10 PCTFULL: Two new display fields added  
NEWS 7 OCT 21 BIOSIS file reloaded and enhanced  
NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced  
NEWS 9 NOV 24 MSDS-CCOHS file reloaded  
NEWS 10 DEC 08 CABA reloaded with left truncation  
NEWS 11 DEC 08 IMS file names changed  
NEWS 12 DEC 09 Experimental property data collected by CAS now available in REGISTRY  
NEWS 13 DEC 09 STN Entry Date available for display in REGISTRY and CACApplus  
NEWS 14 DEC 17 DGENE: Two new display fields added  
NEWS 15 DEC 18 BIOTECHNO no longer updated  
NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer available  
NEWS 17 DEC 22 Additional INPI reactions and pre-1907 documents added to CAS databases  
NEWS 18 DEC 22 IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields  
NEWS 19 DEC 22 ABI-INFORM now available on STN  
NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable  
NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CACApplus  
NEWS 22 FEB 05 German (DE) application and patent publication number format changes  
  
NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
NEWS INTER General Internet Information  
NEWS LOGIN Welcome Banner and News Items  
NEWS PHONE Direct Dial and Telecommunication Network Access to STN  
NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

\*\*\*\*\* STN Columbus \*\*\*\*\*

FILE 'HOME' ENTERED AT 16:51:12 ON 05 FEB 2004

=> FIL BIOSIS EMBASE CAPLUS  
COST IN U.S. DOLLARS SINCE FILE TOTAL  
ENTRY SESSION  
FULL ESTIMATED COST 0.21 0.21

FILE 'BIOSIS' ENTERED AT 16:51:20 ON 05 FEB 2004  
COPYRIGHT (C) 2004 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 16:51:20 ON 05 FEB 2004  
COPYRIGHT (C) 2004 Elsevier Inc. All rights reserved.

FILE 'CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004  
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.  
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.  
COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

=> s pituitary adenylate cyclase activating polypeptide or PACAP  
L1 6494 PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP

=> s l1 and (knockout or transgen? or disrupt? or delet? or deficien?)  
L2 368 L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)

L2 ANSWER 1 OF 368 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2004:13351 BIOSIS  
DN PREV200400012847  
TI \*\*\*PACAP\*\*\* treatment maintains the beta cell mass and retards the onset of hyperglycemia in beta cell-specific calmodulin-overexpressing \*\*\*transgenic\*\*\* mice.  
AU Tsunekawa, S. (Reprint Author); Miura, Y. (Reprint Author); Yamamoto, N. (Reprint Author); Ariyoshi, Y. (Reprint Author); Senda, T.; Oiso, Y. (Reprint Author); Niki, I.  
CS First Dept of Internal Medicine, Nagoya University, School of Medicine, Nagoya, Japan  
SO Diabetes & Metabolism, (August 2003) Vol. 29, No. Hors serie 2, pp. 4S58.  
print  
Meeting Info.: 18th International Diabetes Federation Congress, Paris, France, August 24-29, 2003.  
ISSN: 1262-3636.  
DT Conference; (Meeting)  
Conference; Abstract; (Meeting Abstract)  
LA English  
ED Entered STN: 24 Dec 2003  
Last Updated on STN: 24 Dec 2003

=> d his

(FILE 'HOME' ENTERED AT 16:51:12 ON 05 FEB 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004  
L1 6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP  
L2 368 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)

=> s l1 (1s) (knockout or transgen? or disrupt? or delet? or deficien?)  
L3 280 L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)  
L4 154 DUP REM L3 (126 DUPLICATES REMOVED)

=> dup rem l3  
PROCESSING COMPLETED FOR L3  
L4 154 DUP REM L3 (126 DUPLICATES REMOVED)

=> s l4 and PY=2002  
L5 35 L4 AND PY=2002

=> s l4 and PY<=2002  
2 FILES SEARCHED...  
L6 124 L4 AND PY<=2002

=> d his

(FILE 'HOME' ENTERED AT 16:51:12 ON 05 FEB 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004  
L1 6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP  
L2 368 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)  
L3 280 S L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICIEN?)  
L4 154 DUP REM L3 (126 DUPLICATES REMOVED)  
L5 35 S L4 AND PY=2002  
L6 124 S L4 AND PY<=2002

=> s l2 and psychiatr?  
L7 3 L2 AND PSYCHIATR?

=> dup rem l7  
PROCESSING COMPLETED FOR L7  
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

=> d bib abs 1-  
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):  
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L8 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2003:1004671 CAPLUS  
DN 140:3785  
TI Non-human animal model for \*\*\*psychiatric\*\*\* disorder with \*\*\*deficient\*\*\* in function of \*\*\*pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* - \*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* gene  
IN Baba, Akemichi; Matsuda, Toshio; Hashimoto, Hitoshi; Shintani, Norihito  
PA Japan  
SO U.S. Pat. Appl. Publ., 13 pp., Cont.-in-part of U.S. Pat. Appl. 2001 34,885.  
CODEN: USXXCO  
DT Patent  
LA English

## FAN.CNT 2

PATENT NO. KIND DATE APPLICATION NO. DATE

PI US 2002162128 A1 20021031 US 2002-73135 20020213  
US 2001034885 A1 20011025 US 2001-835627 20010417

PRAI JP 2000-118088 A 20000419  
US 2001-835627 B2 20010417

AB The invention relates to mammalian model animal for \*\*\*psychiatric\*\*\* disorders having a chromosome of a somatic cell and a germ cell with \*\*\*deficiency\*\*\* of function of \*\*\*pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* - \*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* ( \*\*\*PACAP\*\*\* ) gene. The exon 5 of gene \*\*\*PACAP\*\*\* of mammal is \*\*\*disrupted\*\*\* and replaced with neomycin resistance gene. The results of behavioral expts. with \*\*\*PACAP\*\*\* -/- mice demonstrate that \*\*\*disruption\*\*\* of the \*\*\*PACAP\*\*\* gene in mice lead to perturbations in psychomotor behaviors, esp. the exploratory component of locomotor behavior, implicating \*\*\*PACAP\*\*\* in psychotic brain functions. Furthermore, the 5-HIAA level was decreased slightly in the cortex and striatum of the \*\*\*PACAP\*\*\* -/- mouse brain. One of the striking findings of the present study was that \*\*\*PACAP\*\*\* -/- mice showed abnormal jumping behavior in the open field arena. The \*\*\*PACAP\*\*\* -/- mouse should be a valuable tool to investigate both normal and pathol. processes in which \*\*\*PACAP\*\*\* has been proposed to play a role.

L8 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2002:587826 BIOSIS  
DN PREV200200587826

TI Higher brain functions of \*\*\*PACAP\*\*\* and a homologous Drosophila memory gene amnesiac: Insights from knockouts and mutants.

AU Hashimoto, Hitoshi; Shintani, Norihito; Baba, Akemichi [Reprint author]

CS Laboratory of Molecular Neuropharmacology, Graduate School of Pharmaceutical Sciences, Osaka University, 1-6 Yamadaoka, Suita, Osaka, 565-0871, Japan  
baba@phs.osaka-u.ac.jp

SO Biochemical and Biophysical Research Communications, (September 27, 2002)  
Vol. 297, No. 3, pp. 427-432. print.  
CODEN: BBRCA9. ISSN: 0006-291X.

DT Article  
LA English  
ED Entered STN: 13 Nov 2002  
Last Updated on STN: 13 Nov 2002

AB Neuropeptides usually exert a long-lived modulatory effect on the small-molecule neurotransmitters with which they colocalize via regulation of the response times of second messenger systems. \*\*\*Pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* - \*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* ( \*\*\*PACAP\*\*\* ) functions as a neuromodulator and neurotransmitter and regulates a variety of physiological processes. \*\*\*PACAP\*\*\* is structurally highly conserved during evolution, implying its vital importance. In Drosophila, loss-of-function mutations in a \*\*\*PACAP\*\*\* -like neuropeptide gene, amnesiac (amn), affect both memory retention and ethanol sensitivity. The amnesiac gene is expressed in neurons innervating the mushroom body lobes, the olfactory associative learning center. Conditional genetic ablation of neurotransmitter release from these neurons mimics the amnesiac memory phenotypes, suggesting an acute role for amnesiac in memory. However, genetic rescue experiments also suggest developmental defects in amnesiac mutants, implying a role in neuronal development. There is a parallel between memory formation in Drosophila and mammals. \*\*\*PACAP\*\*\* -specific (PAC1) receptor-\*\*\*deficient\*\*\* mice show a deficit in hippocampus-dependent associative learning and mossy fiber long-term potentiation (LTP). Meanwhile, \*\*\*PACAP\*\*\* -\*\*\*deficient\*\*\* mice display a high early mortality rate and additional CNS phenotypes including behavioral and psychological phenotypes (e.g., hyperlocomotion, intense novelty-seeking behavior, and explosive jumping). A functional comparison between \*\*\*PACAP\*\*\* and amnesiac underlines phylogenetically conserved functions across phyla and may provide insights into the possible mechanisms of action and evolution of this neuropeptidergic system.

L8 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 1999:28142 BIOSIS  
DN PREV199900028142

TI Truncated presenilin 2 derived from differentially spliced mRNAs does not affect the ratio of amyloid beta-peptide 1-42/1-40.

AU Gruenberg, Juergen; Walter, Jochen; Eckman, Chris; Capell, Anja; Schindzielorz, Alice; Younkin, Steven; Mehta, Nitin; Hardy, John; Haass, Christian [Reprint author]

CS Central Inst. Mental Health, Dep. Molecular Biol., J5, 68159 Mannheim, Germany

SO Neuroreport, (Oct. 5, 1998) Vol. 9, No. 14, pp. 3293-3299. print.  
CODEN: NERPEZ. ISSN: 0959-4965.

DT Article  
LA English  
ED Entered STN: 3 Feb 1999  
Last Updated on STN: 3 Feb 1999

AB Numerous mutations in the presenilin (PS) genes cause early onset familial Alzheimer's disease (FAD). Here we characterize the expression of two naturally occurring alternative PS2 transcripts which lack either exons 3 and 4 (PS2 DELTAexon3,4) or exons 3, 4, and 8 (PS2 DELTAexon3,4,8). These transcripts do not contain the natural initiation codon within exon 3.

The transcripts are efficiently translated as N-terminal truncated proteins. These \*\*\*deleted\*\*\* proteins are still able to regulate formation of endogenous PS fragments, indicating that the C-terminal half of the PS2-protein is sufficient for this phenomenon. Although approx 50% of the PS1 and both PS2 mutations occur within the N-terminal region lacking in the PS2 DELTAexon3,4 and PS2 DELTAexon3,4,8 proteins, expression of these truncated proteins does not affect pathological generation of amyloid beta-peptide (Abeta). This suggests that point mutations causing AD are gain of function mutations.

&gt;&gt; d his

(FILE 'HOME' ENTERED AT 16:51:12 ON 05 FEB 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004

L1 6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP

L2 366 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICI

L3 280 S L1 (15) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICI

L4 154 DUP REM L3 (126 DUPLICATES REMOVED)

L5 35 S L4 AND PY=2002

L6 124 S L4 AND PY<=2002

L7 3 S L2 AND PSYCHIATRY

L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

&gt;&gt; s l2 and homozygo?

L9 7 L2 AND HOMOZYGO?

&gt;&gt; dup rem l9

PROCESSING COMPLETED FOR L9

L10 4 DUP REM L9 (3 DUPLICATES REMOVED)

&gt;&gt; d bib abs 1-

YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2003:282277 CAPLUS  
DN 138:282471

TI Use of human and mouse insulin 6 gene-encoded protein in improving spermatocyte motility in diagnosis and treatment of male sterility

IN Menon, Ram K.; Sperling, Mark A.; Lu, Chunxia; Witche, Selma; Kasik, John

PA Children's Hospital of Pittsburgh, USA

SO PCT Int. Appl., 92 pp.  
CODEN: PIXXD2

DT Patent  
LA English  
FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2003028457 A1 20030410 WO 2002-US030781 20020927

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MY, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, BG, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003229035 A1 20031211 US 2001-987399 20010928

PRAI US 2001-987399 A 20010928

AB The present invention relates to a novel gene from the insulin family, INSL6, which expresses a protein restoring motility in ciliated cells. The proteins of the insulin family play essential roles in pleiotropic physiol. processes affecting metab., growth, and reprod. A new member of the insulin family named Ins16 is disclosed playing an essential role in ciliated cell activity. Ins16 plays an essential role in spermatocyte function. Thus, the INSL6 gene and its protein product are useful in the treatment of infertility caused by the loss of spermatocyte motility. A method of modulating male fertility is disclosed.

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2001:504687 BIOSIS  
DN PREV200100504687

TI Sympathoadrenal function in \*\*\*pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* - \*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* ( \*\*\*PACAP\*\*\* ) - \*\*\*deficient\*\*\* mice.

AU Hamelink, C. R. [Reprint author]; Lee, H. W. [Reprint author]; Damadzic, R. [Reprint author]; Tjurmina, O.; Young, W. S. [Reprint author]; Weihe, E.; Eiden, L. E. [Reprint author]

CS Lab. of Cellular and Molecular Regulation, NIMH, NIH, Bethesda, MD, USA

SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 620. print.

Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.

ISSN: 0190-5295.

DT Conference; (Meeting)  
Conference; Abstract (Meeting Abstract)  
LA English  
ED Entered STN: 31 Oct 2001

Last Updated on STN: 23 Feb 2002  
AB \*\*\*PACAP\*\*\*'s role as a splanchnic neurotransmitter regulating adrenomedullary secretion is imprecisely defined. We generated ES cells heterozygous for \*\*\*PACAP\*\*\* deletion by homologous recombination, and from them, mice \*\*\*homozygous\*\*\* for the wild-type (+/+) or null (-/-) \*\*\*PACAP\*\*\* allele. Challenge with 2.5 U/kg of insulin resulted in decreased survival, a less profound elevation of circulating epinephrine, and a more profound hypoglycemia, in (-/-) than in (+/+) mice. Decreased survival of (-/-) mice after insulin challenge could be partially reversed by concomitant administration of glucose (20ug/mouse/hour, i.p.), isoproterenol (3ug/mouse/hour, i.p.), or \*\*\*PACAP\*\*\* (10nmol/mouse single dose, i.p.) with 5 U/kg insulin (i.p.). In addition to decreased epinephrine output in \*\*\*PACAP\*\*\* (-/-) mice following insulin, \*\*\*PACAP\*\*\* (-/-) mice exhibited no elevation in the activity of adrenal tyrosine hydroxylase, the rate-limiting enzyme in catecholamine biosynthesis, whereas adrenal tyrosine hydroxylase activity was doubled 4-8 hours after insulin administration (2 U/kg) in \*\*\*PACAP\*\*\* (+/+) mice. These data suggest that \*\*\*PACAP\*\*\* is required to couple secretion and biosynthesis of adrenomedullary catecholamines to maintain plasma catecholamine levels sufficient for gluconeogenesis during prolonged hypoglycemia.

L10 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 1

AN 2000:491720 BIOSIS  
DN PREV200000491841  
TI \*\*\*Pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* .  
\*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* precursor is processed solely by prohormone convertase 4 in the gonads.  
AU Li, Min [Reprint author]; Mbikay, Majambu; Arimura, Akira  
CS U.S.-Japan Biomedical Research Laboratories, Tulane University Hebert Center, 3705 Main Street, Belle Chasse, LA, 70037-3001, USA  
SO Endocrinology, (October, 2000) Vol. 141, No. 10, pp. 3723-3730. print  
CODEN: ENDOAO. ISSN: 0013-7227.

DT Article  
LA English  
ED Entered STN: 15 Nov 2000  
Last Updated on STN: 10 Jan 2002

AB \*\*\*Pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* .  
\*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* (\*\*\*PACAP\*\*\* ) is abundant not only in the brain, but also in the testis. Immunohistochemical studies have shown that \*\*\*PACAP\*\*\* -LI in rat testis is expressed stage specifically in spermatids. This suggests that testicular \*\*\*PACAP\*\*\* participates in the regulatory mechanism of spermatogenesis. Additionally, the ovary contains a relatively small amount of \*\*\*PACAP\*\*\* , conceivably involved in the regulation of folliculogenesis. \*\*\*PACAP\*\*\* is synthesized as a prehormone and is processed by prohormone convertases, such as PC1, PC2, and PC4. PC4 is expressed only in the testis and ovary, where neither PC1 nor PC2 is expressed. However, whether PC4 is the sole endoprotease for the \*\*\*PACAP\*\*\* precursor in the gonads remains unknown. Recent studies using PC4- \*\*\*transgenic\*\*\* mice revealed that male PC4-null mice exhibited severely impaired fertility, although spermatogenesis appeared to be normal. The female PC4-null mice exhibited delayed folliculogenesis in the ovaries. To examine whether PC4 is the sole processing enzyme for the \*\*\*PACAP\*\*\* precursor in the gonads, we analyzed testicular and ovarian extracts from the PC4-null and wild-type mice for \*\*\*PACAP\*\*\* (PACAP38 and PACAP27) and its messenger RNA using reverse phase HPLC combined with specific RIAs and ribonuclease protection assay, respectively. For RIAs, three different polyclonal antisera with different recognition sites were used to identify PACAP38, PACAP27, and its precursor. Neither the testis nor the ovary from the PC4-null mice expressed PACAP38 or PACAP27, but the levels of \*\*\*PACAP\*\*\* transcripts in the testis and ovary of \*\*\*homozygous\*\*\* PC4- \*\*\*deficient\*\*\* mice were considerably elevated compared with those of the wild-type and heterozygous animals. The findings indicate that PC4 is the sole processing enzyme for the precursor of \*\*\*PACAP\*\*\* in the testis and ovary of mice. The possibility that the absence of bioactive \*\*\*PACAP\*\*\* in the testis and ovary of PC4-null mice caused severely impaired fertility in the males and delayed folliculogenesis in females warrants investigation.

L10 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 2

AN 1995:34746 BIOSIS  
DN PREV199598049046  
TI Molecular Basis of Familial Growth Hormone \*\*\*Deficiency\*\*\* .  
AU Perez Jurado, L. A.; Argente, J. [Reprint author]  
CS Div. Paediatr. Endocrinol., Hosp. Nino Jesus, Avda. Menendez y Pelayo, 65, E-28009 Madrid, Spain  
SO Hormone Research (Basel), (1994) Vol. 42, No. 4-5, pp. 189-197.  
CODEN: HRMRA3. ISSN: 0301-0163.

DT Article  
General Review; (Literature Review)  
LA English  
ED Entered STN: 25 Jan 1995  
Last Updated on STN: 14 Mar 1995

AB A significant proportion of cases of GH \*\*\*deficiency\*\*\* (5-30%) may

be due to genetic causes. At least four Mendelian types of isolated GH \*\*\*deficiency\*\*\* (IGHD) have been delineated based on the mode of inheritance and the degree of GH \*\*\*deficiency\*\*\* : IGHD type IA, autosomal recessive with absent endogenous GH; type IB, autosomal recessive with diminished GH; type II, autosomal dominant with diminished GH; and type III, X-linked with diminished GH. Most patients with IGHD type IA have heterogeneous \*\*\*deletions\*\*\* , ranging in size from 6.7 kb to 45 kb, that encompass the entire gene encoding for pituitary GH, GH-1. Nonsense, frameshift and splice GH-1 mutations that predict a complete lack of bioactive GH synthesis in \*\*\*homozygotes\*\*\* have also been reported in association with IGHD IA. Additionally, some cases of IGHD type II have dominant negative mutations in one allele of the GH-1 gene. Panhypopituitary Dwarfism (PD), a condition characterized by \*\*\*deficiency\*\*\* of at least other pituitary trophic hormone in addition to GH \*\*\*deficiency\*\*\* , can have autosomal and X-linked modes of inheritance. Interestingly, both recessive and dominant mutations at the gene encoding for the pituitary transcription factor Pit-1 have been found in a specific subtype of PD that combines GH, prolactin and TSH \*\*\*deficiencies\*\*\* . In contrast, the loci and mutations responsible for the other Mendelian forms of IGHD and PD remain unknown. Linkage studies using genetic markers have excluded the GH locus on chromosome 17 in approx 50% of the cases and the GH-releasing hormone (GHRH) locus on chromosome

20 in all the studied families (types IB and II) in whom the mutation cannot be traced to defects in these genes. Furthermore, several uncharacterized loci on the X chromosome must be required for normal GH secretion. In summary, genetic studies have provided a better understanding of the mechanism of GH \*\*\*deficiency\*\*\* as well as new tools for specific diagnosis of several forms of IGHD and PD. However, isolation and evaluation of other genes involved in GH secretion is still necessary. Several possible candidate genes have been recently cloned and characterized, including genes encoding the human GHRH receptor, the \*\*\*pituitary\*\*\* \*\*\*adenylate\*\*\* \*\*\*cyclase\*\*\* \*\*\*activating\*\*\* \*\*\*polypeptide\*\*\* (\*\*\*PACAP\*\*\* ) and the \*\*\*PACAP\*\*\* receptor. Analysis of these genes in IGHD and PD families may clarify the molecular basis of the defect and also provide new insights into the complex regulation of GH.

=> FIL STNGUIDE  
COST IN U.S. DOLLARS  
ENTRY SINCE FILE TOTAL  
FULL ESTIMATED COST 69.07 69.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE  
TOTAL  
ENTRY SESSION  
CA SUBSCRIBER PRICE -1.39 -1.39

FILE 'STNGUIDE' ENTERED AT 17:01:45 ON 05 FEB 2004  
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT  
COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE  
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM  
KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.  
LAST RELOADED: Jan 30, 2004 (20040130/UP).

=>  
---Logging off of STN---

=>  
Executing the logoff script..

=> LOG Y

COST IN U.S. DOLLARS  
ENTRY SINCE FILE TOTAL  
FULL ESTIMATED COST 0.06 69.34  
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE  
TOTAL

ENTRY SESSION  
CA SUBSCRIBER PRICE 0.00 -1.39  
STN INTERNATIONAL LOGOFF AT 17:02:04 ON 05 FEB 2004